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Antioxidants and nutrigenomics in periodontics: Decoding the futuristic aspect in periodontal care

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Abstract
Excessive presence of free radicals due to oxidative stress or antioxidant deficiency can result in increased levels of Reactive Oxygen Species (ROS) and reactive nitrogen species (RNS), resulting in various inflammatory pathways leading to periodontal destruction. Antioxidants can be used as adjuncts as they act via signal transduction pathways for the management of periodontitis, however the resources on genetic implication of antioxidants is limited. Nutrigenomics, an emerging branch of science, can be implicated in the study of how dietary antioxidant affects the periodontium on a genetic level. This review aims to highlight the link between dietary antioxidants and periodontitis on a genetic level.

Keywords: Nutrigenomics, periodontitis, antioxidants, diet, gene, epigenetics

Introduction
Periodontitis is a chronic, inflammatory, multifactorial disease that results in the destruction of the supporting structures of the teeth, including, gingiva, periodontal ligament, alveolar bone and cementum [1, 2].
Periodontitis has been linked to a number of contributing factors, such as smoking, environmental factors, hereditary factors, and nutritional factors [3]. Given that nutrition has been linked to a number of conditions, including diabetes, rheumatoid arthritis, and cardiovascular illnesses, which have been shown to be closely linked to periodontitis, it may be crucial in regulating and managing the condition [4].
Periodontitis, although initiated by biofilm accumulation, results in the further destruction as a result of various inflammatory mechanisms that generates pro-inflammatory mediators and reactive oxygen species (ROS) [7]. Oxidative stress is an important pathogenic factor for periodontitis and it results due to an imbalance between the production and accumulation of reactive species. Antioxidants therefore play an important role in controlling the destructive progression of periodontitis by inhibiting the ROS.
Diet has been shown to have a role on the human genome by acting directly or indirectly. Thereby, studying the relationship of nutrient on the genome can help us in the prevention and treatment of periodontitis [5]. Various macro and micro nutrients have been advocated as an adjunct for the treatment of periodontitis of which antioxidant supplementation plays a major role. Nutrigenomics is a branch of science that deals with the relationship between nutrition and the genome, thereby facilitating dietary interventions for the prevention and management of diseases [6]. It aims to understand the diet- gene interactions and apply it for the nutritional research for periodontitis patients.

History - background and origin of nutrigenomics
- 25th April 1953, Watson and Crick printed “the molecular structure of DNA”.
- 1997, the first nutrigenomics company was started.
- 1999, the name nutritional genomics was proposed as genomics by Nancy Fogg-Johnson and Alex Merolli which provides strong means of discovering hereditary factors in disease.
- Pelegrin originally used the word "nutrigenomics" in 2001, and Van Ommen and Stierum used it in a review in 2002.

- The Human Genome Project, which included the full sequencing of the human genome and was started on April 14, 2003, with the help of former US President Bill Clinton and former British Prime Minister Tony Blair, was seen as the beginning of the genomic age.
- The European Nutrigenomics Organization, or NuGo, was founded in 2004 and received funding through June 2010.
- In 2007, the Kluyver Centre for Genomics of Industrial Fermentation in the Netherlands partnered with the Nestle Research Center.
- A US Berkeley scientist forecasted a human genome test within five years in 2008.

What is nutrigenomics?

Nutrigenomics, a constituent of nutritional genomics takes into consideration the effect of food on gene expression [8].

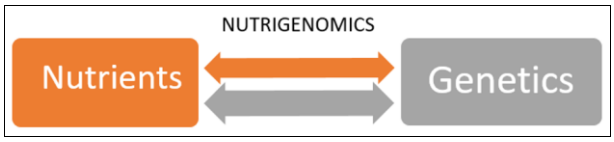

Fig 1: The Relationship Between Nutrients and Genetics in Nutrigenomics

The study of nutrigenomics is heavily reliant on the new and improved technology that enable us to process vast amounts of information on gene variants. These so-called "omic" technologies-genomic, proteomic, metabolomics, and transcriptomic-allow us to simultaneously identify and test a wide variety of molecules.

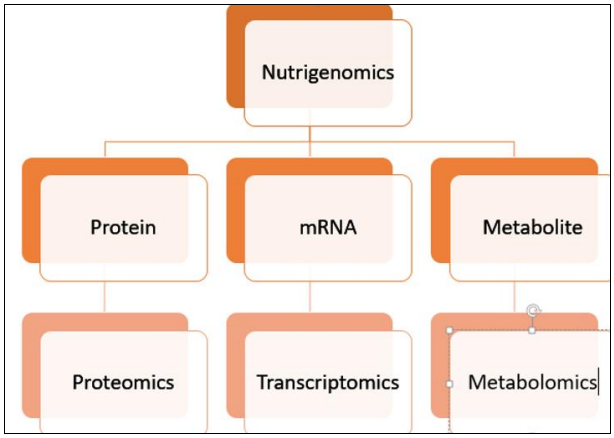

Fig 2: The Integration of Multi-Omics Technologies in Nutrigenomics

Mechanism of action of nutrigenomics

Cells are always evolving and adjusting to their surroundings, thereby a regulated system that handles the presence of nutrients or food/dietary components, infections, or harmful components from the absorption process is maintained. The dietary components and food that are absorbed act as dietary cues. These signals either directly or indirectly affect how transcription factors, which are intracellular proteins, function [9].

Nutrients may

1. Directly bind to transcription factor receptors at the cellular level
2. May change the concentration of substrates involved in gene regulation or cell signalling by being absorbed by primary or secondary metabolic pathways.
3. Modify the pathways involved in signal transduction the nutrients attach to transcription factors in the case of direct regulation. The primary mechanisms by which nutrients affect gene expression are transcription factors [9-12].

Epigenetics and Its Impact on Periodontal Health

The term epigenetics describes the interaction between epigenetic modification and the control of gene expression and differentiation, as well as genetic alterations in cell phenotypic or gene activity that take place without altering the DNA sequence [13].

Regulating gene expression during growth, development, aging, and the onset of illness is mostly accomplished by epigenetic control. Effective operation of the epigenetic regulatory network is crucial for an individual's life to respond to environmental influences in an orderly manner [13].

The primary epigenetic mechanisms are histone modification, DNA methylation, non-coding RNA molecules, and the regulation of gene expression by mRNA methylation. Cells can transfer genetic information and associated phenotypes through the cooperation of many epigenetic regulators in response to developmental or environmental stimuli [14-16].

Oxidative stress and Periodontitis

Growing research in recent years has demonstrated the critical role oxidative stress plays in the pathophysiology of periodontitis and other forms of chronic inflammation. Periodontal tissue loss can be decreased by preventing OS-gene activation in inflammation, which protects periodontal tissues or cells from oxidative damage [3].

Sies originally defined oxidative stress as "a process in which the balance between oxidants and antioxidants is shifted toward the oxidant side" (1985, 1986). The pathophysiology of cardiovascular illness (Siekmeier *et al*. 2007), type 2 diabetes (Allen *et al*. 2009), obesity/metabolic dysregulation (Bullon *et al*. 2009), and periodontitis (Chapple & Matthews 2007) is all supported by oxidative stress. It has been proposed that the pro-inflammatory cascades that underlie tissue damage in the inflammatory states linked to periodontitis are primarily orchestrated by oxidative stress (Chapple 2009).

During regular metabolism, electrons may escape from the inner mitochondrial membrane at complex III of the hydrogen-electron transport chain, which can lead to oxidative stress (Battino *et al*. 1999). This results in the formation of the superoxide radical, which is the single electron reduction of molecular oxygen. In this case, consuming more refined sugars or saturated fats (which are macronutrients) can overload the Krebs cycle, resulting in reactive oxygen species (ROS) and superoxide radicals. These ROS overwhelm the antioxidant defense systems of the mitochondria (superoxide dismutase 2), causing oxidative stress.

Nuclear factor kappa B (NFkB) and activating protein-1 (AP-1) are two examples of redox sensitive gene transcription factors that are activated by oxidative stress and have a pro-inflammatory function. These transcription factors have several redox-regulated activation cascade points, and the generation of pro-inflammatory cytokines can be triggered by the depletion of intracellular antioxidants such Glutathione (GSH) (Chapple 1996). Oxidized LDL, AGE-RAGE interactions, ROS production, and pro-inflammatory cytokines are all factors that can activate NFkB.

Numerous signaling pathways, including the NF-κB, IL-17, and Wnt/β-catenin signaling pathways, are implicated in the development of periodontitis [30]. NF-κB signaling has a role in the osteogenesis of periodontal ligament stem cells after inflammatory activation [31]. The metabolism of alveolar bone is associated with the IL-17 signaling system. According to GSEA data, the OS-genes in periodontitis primarily contributed to the IL-17 signaling pathway, osteoclast differentiation, and leukocyte transendothelial migration [30].
CXCR4 is a critical mediator of proteinuria, glomerulosclerotic lesions, and podocyte damage brought on by oxidative stress. Alveolar bone resorption has been demonstrated to be markedly inhibited by CXCR4 neutralization in periodontal inflammation.In periodontitis, CXCR4 modifies the mechanical sensitivity of the periodontal tissue and can prevent the release of nitric oxide from invading macrophages. One of the major OS-genes that was identified to be most overexpressed in periodontitis tissues was CXCR4. Chemokine receptor activity, chemokine responsiveness, C-C chemokine receptor activity, and C-C chemokine binding may all be regulated by it. Consequently, it is reasonable to hypothesize that the chemotactic activity could explain how CXCR4 contributes to the development of periodontitis [30].
NFkB and downstream cytokine production can be down-regulated by raising intracellular GSH concentrations, or the GSH: GSSG ratio (a measure of intracellular redox status and antioxidant levels; GSSG is the oxidized version of GSH and a non-radical) (Rahman *et al*. 2005).

Effects of reactive oxygen species on periodontal tissues and components:
Damage to periodontal tissue is caused by ROS through:

- Degradation of ground substances
- Direct or indirect collagenolysis
- Collagenolysis due to protease oxidation
- Activation of nuclear factor-κB (NF-κB) to stimulate the release of excessive pro-inflammatory cytokines
- Superoxide release and lipid peroxidation, which have both been connected to bone resorption, produce PG-E2.
- The combined effects of endotoxin-mediated cytokine production and that resulting from respiratory burst of polymorphonuclear leukocytes in response to the same organisms cause periodontal inflammation and subsequent attachment loss because interleukin-1 (IL-1) and tumour necrosis factor alpha positively regulate their own production [17].

An oxidative stress response, thought to be connected to periodontal damage, can be triggered by a homeostatic imbalance between ROS and antioxidant defense mechanisms (Waddington *et al*., 2000; Baltacioglu *et al*., 2014b). Malondialdehyde (MDA) and total oxidant status (TOS) levels are clinically strongly positively correlated with periodontal parameters (Akalin *et al*., 2007; Baltacioglu *et al*., 2014b) [32].
Reduced ROS levels reduced bone loss by downregulating the expression of genes that indicate osteoclast development (Kanzaki *et al*., 2013). Through redox-sensitive gene transcription factors like nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB), ROS can also trigger immunological responses (Gan *et al*., 2016). Furthermore, via activating c-Jun N-terminal kinase (JNK), ROS can cause cellular death (Liu *et al*., 2015) [32].

How oxidants and antioxidants affect the gene expression
Cellular oxidant/ antioxidant equilibrium is a key determinant in redox dependent signal transduction pathways both *in-vivo* and *in-vitro*. Antioxidant nutrients modulate key enzymes such as phosphatases and kinases by interacting with cell receptors. Changes in the transcription factor levels results in changes in mRNA and proteins. Antioxidants through their protein binding activity can directly act on the enzymes and change their activity [18].

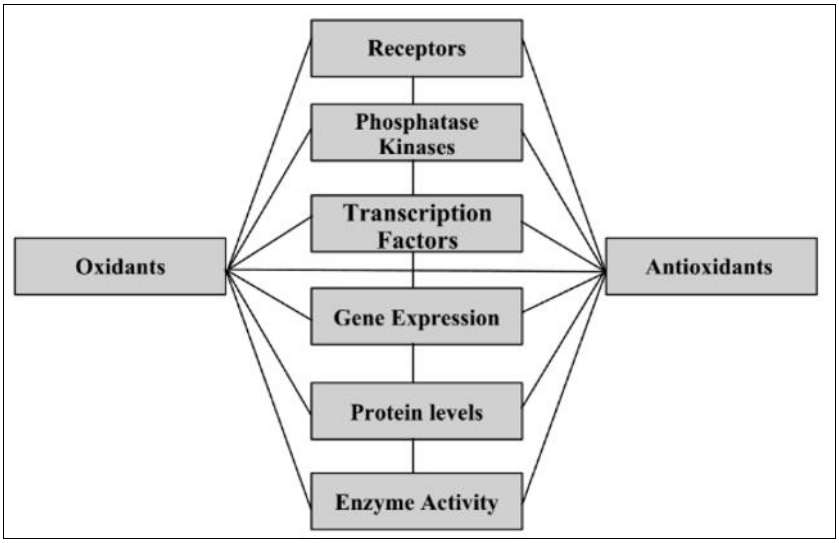

Fig 3: Cell receptors, cellular key enzymes, and transcription factors as molecular targets of oxidants and antioxidants.

Thereby, antioxidants in addition to being scavengers for ROS and RNS and preventing oxidative damage towards lipids, proteins and DNA, also act as cell signalling molecules and their combined effect has proven beneficial for various chronic inflammatory conditions including, periodontitis [18].
Different transcription factors like NF-ϰB, AP-1, Nrf-1, SP-1 are regulated by the cellular redox status. NF-ϰB is responsible for controlling various genes involved in the inflammatory and proliferative responses [18]. Different transcriptomics methods have been applied for the quantitative and comprehensive analysis of changes in mRNA expressions:

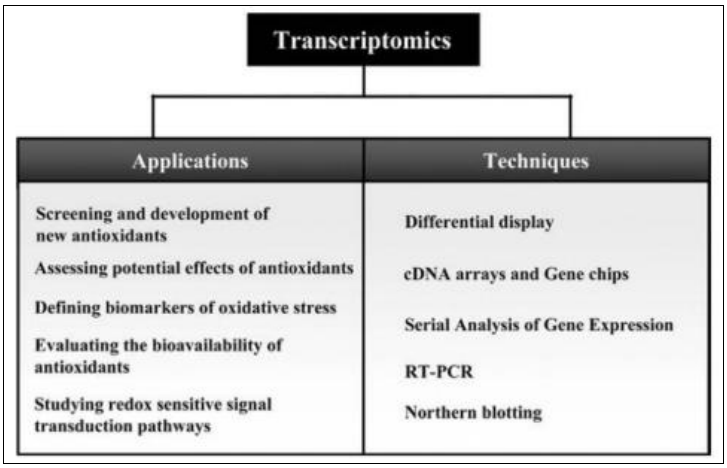

Fig 4: Analytical techniques and potential applications of transcriptomics in the field of free-radical research.

Gene arrays are used for assessing the molecular basis of oxidants and antioxidants, as a biomarker of oxidative stress, to check potency and bioavailability of different antioxidants and to study the toxicity of upcoming novel antioxidants. It can be used by:

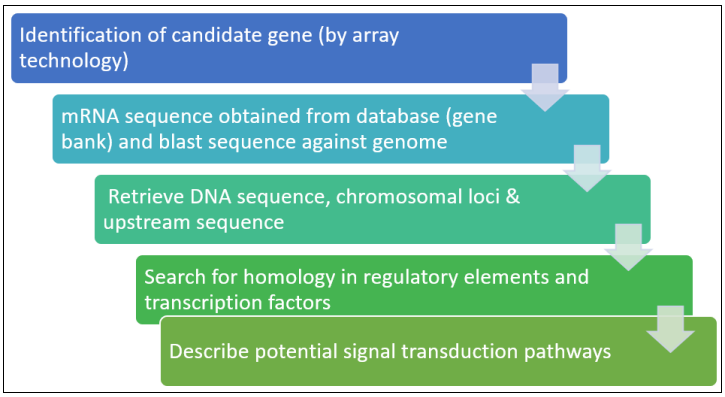

Fig 5: Bioinformatics pipeline for the identification and characterization of candidate genes using array technology and genomic analysis

Thorough research designs and statistical analysis are required to have a thorough grasp of the molecular mechanisms of action of oxidants and antioxidants. To determine whether variations in the gene expression profile are consistently seen over an extended period of time, time-point measurements must be implemented. Differential changes in gene expression in response to dietary interventions were frequently observed in pooled samples and at a single time point in earlier research. Additionally, variations in gene expression levels detected by gene chips technology should always be validated by separate techniques like northern blotting and real-time PCR before being supported by functional parameters [18].

The analytical steps involved in gene chip experiment included

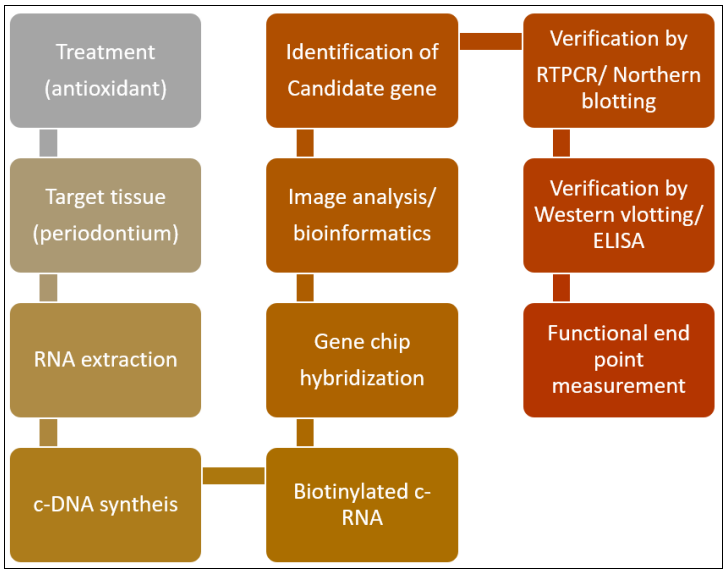

Fig 6: Gene Expression Profiling Workflow tion of for Antioxidant Treatment in Periodontium

Antioxidant and Periodontitis

According to Halliwell and Gutteridge (1989), an antioxidant is "a substance which when present at low concentrations, compared with those of an oxidizable substrate will significantly delay or inhibit oxidation of that substrate.". If the body doesn't have enough antioxidant reserves to make up for the antioxidants that are ingested, this change may result in biological harm as well as antioxidant depletion.

The host defense cells (phagocytic lymphocytes) that generate oxygen radicals during an inflammatory response after being stimulated by opsonized particles, bacterial DNA, or peptides, as well as by activating the hexose-monophosphate shunt, which uses molecular oxygen and NADPH as electron donors, are another endogenous source of oxygen radical formation (Waddington *et al*. 2000, Chapple & Matthews 2007). The enzyme produces superoxide (O2-), which is the precursor of a wide range of reactive oxygen species (ROS), such as singlet oxygen, free radicals, and oxidized halogens. Although phagocytes use these oxidants to eliminate invasive germs, their production must be strictly controlled to ensure that they are only produced when and where needed because they also seriously harm neighboring tissues (Babior 1999) [19].

External stresses include illness, some medications, heat, trauma, ultrasound, UV light, ozone, exhaust fumes, radiation, extreme exercise, and smoking are additional causes of ROS and antioxidant depletion.

Traditionally, many biological compounds have "antioxidant capacities." Vitamin C, vitamin E (tocopherol), carotenoids (α and β-carotene, beta-cryptoxanthin, and zeoxanthin), polyphenols, bilirubin, GSH, uric acid, and melatonin are some that have been researched in relation to periodontal disease, although just a few do. Omega-3 PUFAs and the vitamin B group (niacin (B3), folate, or folic acid (B9)) are examples of non-antioxidant micronutrients that have also been studied.

In order to counteract oxidative stress and pro-inflammatory events, antioxidants:

- Inhibit lipid peroxidation and its by-products (like oxLDL formation)
- Eliminate ROS, which stops AP-1 and NF-κB from activating
- Activate transcription factors for anti-inflammatory genes (like nuclear erythroid2 p45-related factor2).
- Enhancing insulin sensitivity, thereby eliminating the effects of elevated blood and tissue glucose levels
- Avoiding direct harm to proteins (such as α1-antitrypsin oxidation) and DNA [20].

Locally in the periodontium and in plasma, researchers have shown antioxidant depletion in periodontitis. They also discovered an inverse connection between a higher frequency of periodontitis and lower levels of vitamin C and plasma total antioxidants.

Furthermore, reduced glutathione (GSH), the primary intracellular antioxidant redox-regulator of NF-κB, has been suggested by researchers as a potential strategy for downregulating hyperinflammatory processes. In periodontitis, GSH levels seem to be reduced, hence techniques to increase intracellular GSH may be helpful [21].

Linden *et al*. (2009) studied the relationship between periodontal health and antioxidant levels in a Northern Ireland population of 1258 men aged 60-70 years. The study found that levels of α- and β-carotene, β-cryptoxanthin, and zeaxanthin were significantly lower in the moderate and generalized severe periodontitis group compared to the remaining population. However, no significant differences were found in the levels of lutein, lycopene, α- and γ-tocopherol, or retinol. The study also found an inverse relationship between α- and β-carotene and β-cryptoxanthin and moderate periodontitis. However, β-carotene and β-cryptoxanthin were the only antioxidants associated with an increased risk of generalized severe periodontitis.

Antioxidants and Periodontal Health

The oral cavity experiences inflammation and damage from illness and trauma, just like any other tissue. The generation of ROS by immune cells in response to pathogen stimulation is a component of inflammatory processes. The chemical structure of molecules can be changed by ROS and highly reactive free radicals (molecules with unpaired electrons), which can cause harm to cells and tissues. By starting a chain reaction known as lipid peroxidation, they specifically harm lipids [22].

Normally, ROS are produced as a result of aerobic respiration. However, in order to minimize cellular damage, the antioxidant defense enzymes lower the ROS. The antioxidant system, however, is unable to reduce oxidative damage if inflammation or tissue injury results in an excessive production of ROS. Oxidative stress is the result of a disturbance in the equilibrium between the production of ROS and antioxidant enzymes, such as glutathione. Additionally, the development of inflammation brought on by illness and/or trauma can lead the periodontium to enter a state of oxidative stress [23].

In a study done by Alkadasi *et al*, he found that the use of adjunctive *N*-Acetylcysteine (NAC) (600 mg) resulted in a significant reduction in probing depths in the S-NAC (surgey and NAC supplementation) group when compared to the S-non NAC (surgery and no NAC) group at 3 months, but no statistically significant differences in GCF s-RANKL levels were observed in the sites that underwent surgical treatment with or without NAC at different time intervals [24].

Belludi *et al*. (2013) showed that Lycopene (4 mg/day) is a promising treatment modality as an adjunct to full-mouth SRP of the oral cavity in patients with moderate periodontal disease [25].

Elgendy *et al*. (2013) prepared a gel from tea tree oil (TTO) (5%, Melaleuca alternifolia) and concluded that the local delivery of TTO gel in case of chronic periodontitis may have some beneficial effects to the conventional periodontal therapy [26].

Chopra *et al*. (2016) showed that Green tea (Lahijan green tea) reduced Pocket depth and Bleeding index significantly in both groups before and after SRP; this reduction in the intervention group was higher than the control group [27].

Marawar *et al*. (2014), in a clinical trial on melatonin (3 mg daily at night) inferred that there was significant improvement in all the indices in the test group as compared to the control group. Melatonin is a potential antioxidant and the clinical improvement it showed was significantly superior to that of the standard control group [28]

Singh *et al*. (2014) in his study on Vitamin E (200mg every other day) as an antioxidant concluded that Systemic and local Superoxide dismutase levels are lowered in periodontitis and adjunctive vitamin E supplementation improves periodontal healing as well as antioxidant defense [29].

Conclusion

Although some research indicates links between specific

nutrients and genes related to oxidative stress and inflammation, it is difficult to draw firm conclusions or apply findings in clinical settings due to the dearth of extensive, long-term, and interventional studies. More thorough investigation is required to elucidate molecular pathways and ascertain whether antioxidant supplementation based on a person's genetic profile could successfully prevent or treat periodontitis.The study of how different nutrients interact with the genome to affect gene expression, or nutrigenomics, provides a fresh viewpoint on how food affects the immunological and inflammatory responses linked to periodontal disease. However, the existing body of research on nutrigenomics and periodontitis is restricted to *in vitro* or preliminary observational studies and is wide scope yet to be exploited in periodontal research.

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